CASE STUDIES

GAVE: An interesting cause of iron deficiency anemia

Sham Santhanam[1]*, M Madeshwaran[2], Rajeswari Sankaralingam[3]

[1]Postgraduate, Department of Rheumatology, Madras Medical College, Chennai, India
[2]Department of Rheumatology, Madras Medical College, Chennai, India
[3]Professor & HOD, Department of Rheumatology, Madras Medical College, Chennai, India

Abstract

Gastric antral vascular ectasia (GAVE) is a rare cause for chronic severe gastrointestinal bleeding requiring repeated transfusions. We present here the case of 55-year-old female who presented with severe iron deficiency anemia with melena. The disease was further diagnosed as GAVE due to the presence of watermelon stomach on endoscopy with features of limited scleroderma. The patient showed symptomatic improvement on treatment with Argon laser photocoagulation and blood transfusion.

Case report

A 55-year-old female presented to the OPD clinic with dyspnea on exertion, easy fatigability, and melena. She was referred from hematology department due to the occurrence of persistent anemia. No history of hematemesis, abdominal pain or chronic drug intake was reported. She had difficulty in getting up from sitting position, multiple joint pain, and Raynaud's phenomenon. She had been transfused twice for anemia in the last one year. She had attained menopause and had normal bowel habits.

Physical examination revealed that the patient was severely pale with sclerodactyly and facial skin tightening. Her BP was 160/100 mm Hg. She had symmetrical proximal muscle weakness of both lower and upper limbs (power of 3/5). No skin lesions suggestive of dermatomyositis were reported. Other systems examinations were within normal limits.

Her hemoglobin was 5.6 g/dl with normal total count and platelets. Peripheral smear showed microcytic hypochromic anemia and the stool examination was positive for occult blood. Results of upper gastrointestinal (UGI) endoscopy showed hyperemic streaks alternating with normal mucosa (watermelon stomach) (Fig. 1 and 2), suggestive of gastric antral vascular ectasia (GAVE). Her creatine phosphokinase (CPK) was 126 IU/L and the levels of other enzymes such as LDH, SGOT, and SGPT were normal. The findings of electromyography (EMG) were normal with no feature suggestive of inflammatory myositis or myopathy. She was positive for ANA by ELISA and for CENP-B (Anti Centromere Ab) by immunoblot. The patient had undergone Argon laser photocoagulation (Fig. 3 and 4) and packed cells transfusion, and the treatment had contributed to symptomatic improvement.

Based on the clinical investigations and history, she was diagnosed to have limited cutaneous systemic sclerosis with GAVE. The initial presentation of severe iron deficiency anemia was due to intermittent GI bleeding.

Discussion

GAVE is a rare cause for upper GI loss and subsequent iron deficiency anemia. The syndrome was first described by Ryder *et al.* in 1953 and the term 'watermelon stomach' (endoscopic appearance) was first coined by Jabbari *et al.*[1, 2, 3] The disease is under-recognized many a times and misdiagnosed as antral gastritis.[4] It is more common in elderly females and usually diagnosed during endoscopy as prominent flat or raised erythematous patches radiating in a spoke-like fashion from the pylorus to the antrum. The three macroscopic patterns for GAVE disease described by Chawla *et al*. are watermelon stomach, honeycomb stomach, and well-demarcated, round- or mushroom-shaped lesion.[5]

The histology characteristics involve fibromuscular

hyperplasia of lamina propria with intravascular fibrin thrombi and increase in luminal size of mucosal vessels.[4] Although several theories including achlorhydria, hypergastrinemia, and low pepsinogen levels have been proposed; the pathophysiology of GAVE remains unclear.[8] One of the pathogenetic mechanisms proposed is partial prolapse of loosely attached gastric mucosa of antrum due to vigorous gastric peristalsis. So there is intermittent obstruction of submucosal blood vessels and hence vascular ectasia.[2, 4]

The etiology of GAVE is unknown, but the disease is associated with liver cirrhosis (in 30% of patients), systemic sclerosis, primary biliary cirrhosis, chronic renal failure, atrophic gastritis, diabetes mellitus, Addison's disease, familial Mediterranean fever, rectal vascular ectasia, marrow transplantation, and hypothyroidism.[4, 6]

The closest differential diagnosis of GAVE is portal gastropathy. The disease is commonly found in antrum (rather than fundus in portal gastropathy) with no relation to portal hypertension and no response to beta blockers.[9]

el-Omar *et al.* have reported that the association between severe bleeding from GAVE in patients with CREST syndrome is very rare.[10] In Scleroderma: Cyclophosphamide Or Transplant (SCOT) trial, 23 out of 103 (22.3%) patients with early diffuse systemic sclerosis were found to have

Fig. 1 and 2: Gastric mucosa with hyperemic streaks

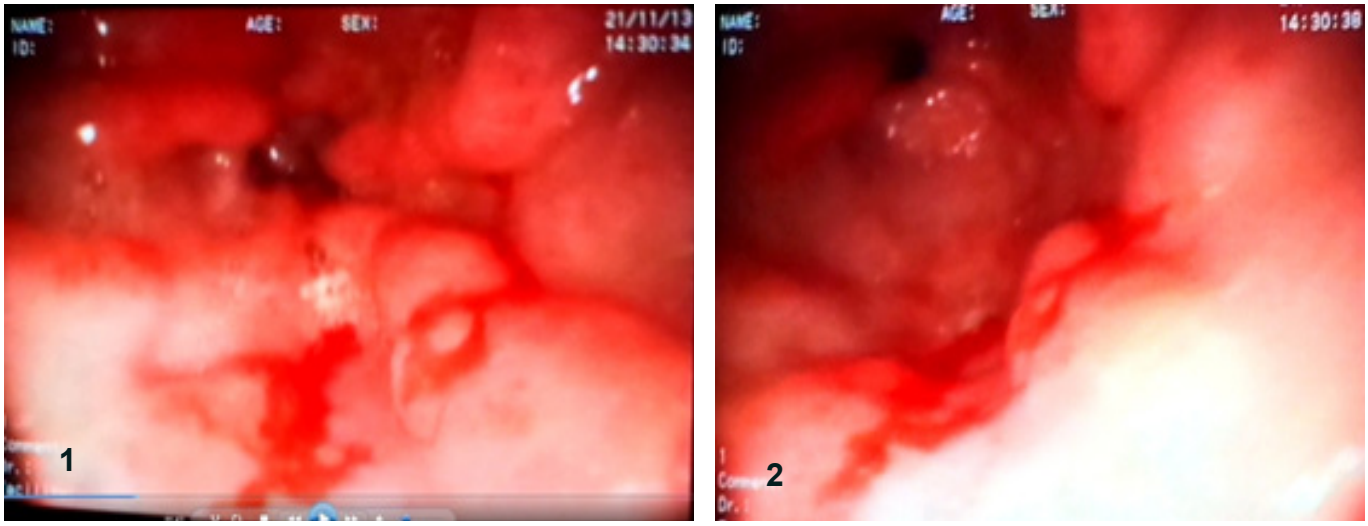
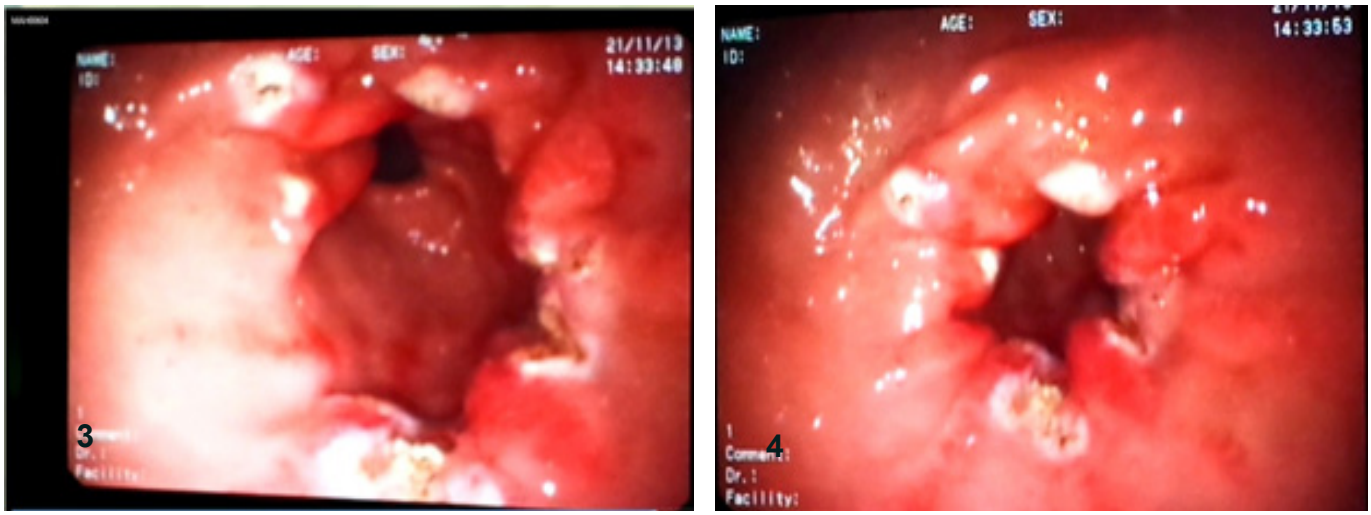


Fig. 3 and 4: Gastric mucosa after Argon laser treatment



GAVE on endoscopy.[11] Anti-topoisomerase I (anti-Scl70) was less frequently associated with GAVE, whereas a negative association between anti-U1 ribonucleoprotein (RNP) antibodies and the disease has been reported.[11] The present case had chronic bleeding requiring multiple transfusions, limited scleroderma, and positive centromere protein (CENP)-B antibodies.

Treatment is often challenging and involves corticosteroids, hormonal therapy, octreotide, and tranexamic acid. Literature evidence also indicates the use of alpha-interferon, calcitonin, serotonin antagonist, and cyproheptadine for treatment. Surgical procedures like antrectomy and shunt procedures may be beneficial in cirrhotic patients. The various endoscopic methods employed are Nd:YAG laser, Argon plasma coagulation, heater probe, sclerotherapy, Argon laser, and bipolar electro-cautery.[4]

Competing interests

The authors declare that they have no competing interests.

Citation

Santhanam S, Madeshwaran M, Sankaralingam R. GAVE: An interesting cause of iron deficiency anemia. IJRCI. 2015;3(1):CS2.

Received: 4 October 2014, **Accepted:** 1 December 2014, **Published:** 9 January 2015

* Correspondence: Dr.Sham S, Postgraduate, Department of Rheumatology, Madras Medical College, Chennai, India
itsdrsham@gmail.com

References

1. Ryder JA, Klotz AP, Kirsner JB. Gastritis with veno capillary ectasia as a source of massive gastric haemorrhage. Gastroenterology. 1953; 24: 118–23.
2. Jabbari M, Cherry R, Lough JO, et al. Gastric antral vascular ectasia: the watermelon stomach. Gastroenterology. 1984; 87: 1165–70.
3. Gardiner GW, Murray D, Prokipachuk EJ. Watermelon stomach, or antral gastritis. J Clin Pathol. 1985; 85: 1317–8.
4. Sebastian S, O'Morain CA, Buckley MJM. Review article: current therapeutic options for gastric antral vascular ectasia. Aliment Pharmacol Ther. 2003 Jul 15;18(2):157–65.
5. Chawla SK, Ramani K, Loaesti P. The honeycomb stomach: coalesced gastric angiodysplasia. Gastrointest Endosc 1990;36: 516–8.
6. Ward EM, Raimondo M, Rosser BG, et al. Prevalence and natural history of gastric antral vascular ectasia in patients undergoing orthotopic liver transplantation. J Clin Gastroenterol 2004;38:898–900.
7. Gostout CJ, Viggiano TR, Ahlquist DA, et al. The clinical and endoscopic spectrum of the watermelon stomach. J Clin Gastroenterol 1992;15:256–263.
8. Kar P, Mitra S, Resnick JM, Torbey CF. Gastric antral vascular ectasia: case report and review of the literature. Clin Med Res. 2013;11(2):80-85.
9. Amarapurkar DN, Patel ND. Gastric antral vascular ectasia (GAVE) syndrome. J Assoc Physicians India. 2004;52:756-758.
10. el-Omar MM, Jenkins AP, Hollowood K, Banerjee AK, Thompson RP. Gastric telangiectasis: a rare cause of severe blood loss in CREST syndrome. Postgrad Med J. 1994;70(822):302-304.
11. Hung EW, Mayes MD, Sharif R, et al. Gastric antral vascular ectasia and its clinical correlates in patients with early diffuse systemic sclerosis in the SCOT trial. J Rheumatol. 2013;40(4):455-460.